MORTALITY AMONG EMPLOYEES OF THE BENDIX AUTOMOTIVE CORPORATION WINDSOR, ONTARIO

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EXECUTIVE SUMMARY

The Ontario Ministry of Labour has carried out an analysis of the mortality pattern of former employees of the Bendix Automotive Corporation in Windsor. This study was conducted in response to concerns, expressed in a letter to the Minister of Labour by the United Auto Workers (now CAW), that the health of employees had been damaged by exposure to asbestos at the Argyle Road and Prince Road factories.

The aim of this study was to investigate whether the exposures at the Windsor factories were associated with an increased risk of death from asbestos-related diseases. This report presents the results of a study of mortality among 1657 workers who were employed at Bendix for one year or more. Deaths occurring between January 1, 1950 and December 31, 1985 were ascertained by a computerized linkage with the Canadian Mortality Data Base at Statistics Canada and form the basis for the analysis.

Bendix Automotive of Canada Limited (BAC) began manufacturing operations in Windsor in 1929 when it purchased property on Argyle Road. In 1940 BAC purchased drilling, grinding, and riveting equipment to manufacture its own lined brake shoes, and it is from this time that employees may have been exposed to asbestos-containing dusts. The type of asbestos fibre contained in the brake linings was chrysotile asbestos and the asbestos content generally varied from 25% to 50% depending upon the particular use for the product. The balance of the linings consisted of a resin binder and various modifiers and fillers. In July, 1963 the entire brake assembly operation was transferred to a new BAC plant located on Prince Road.

There were only 2 small production areas in the 2 plants that contained friction material products. The 2 departments in question were designated with Department numbers 20, 25, and 27 (Department 20 was renamed Department 25). Employment in

the friction materials departments was relatively low. The plant reached its highest hourly rated employment level of 778 employees in 1976 at which time only 88 employees worked in friction material operations.

Although workers who were employed in operations involving friction material brake linings containing asbestos had the greatest potential for exposure to asbestos, most of the other workers had potential for exposure to fibres which may have been distributed to other areas of the plant. The level of exposure outside the friction material areas was presumably lower than the levels within them.

The study cohort consists of all workers who were employed on or after January 1, 1950 for a period of at least 12 months. Employee identifying information was abstracted from plant personnel records in Windsor. Collection of information about the jobs performed by each worker was complicated by the fact that detailed personnel folders were not available for all workers. Detailed work histories were sought later, following ascertainment of mortality, as part of a case-control study of those causes of death potentially associated with exposure to asbestos.

The study population consists of 1657 individuals; 1314 males and 343 females. Less than half (701 or 42%) of the 1657 were hired before 1966 and could thus have had 20 or more years pass between the date of hire and the end of observation on December 31, 1985. Forty-five percent of the cohort were employed less than 5 years, 17 percent were employed between 5 and 10 years, and 38 percent were employed for 10 years or more.

The finding indicate that mortality rates among the men were similar to the Ontario average during the first 30 years from potential first exposure to asbestos. Subsequently, mortality rates were 25% higher than the Ontario rates. Excess mortality during this latter period is attributable to a cluster of 3 deaths from laryngeal cancer and a 30% increase in

mortality rates from circulatory diseases. The mortality pattern among the women is unusual, in that there is no occurrence of the so-called "healthy-worker effect", the common observation that employed populations usually have lower mortality rates than the general population in the first years after the start of employment. The women had elevated mortality rates during all the time intervals under consideration. Overall, there was a small increase in cancer mortality, 9 deaths occurred versus 6.7 expected, but no cancer site occurred more than once. There was a larger elevation in death rates from circulatory diseases, 10 deaths occurred while 5.7 were expected based on Ontario rates.

Mortality rates from circulatory diseases were elevated in both sexes, and there was no trend with time. Overall, there was a 19% increase in circulatory disease mortality in comparison with Provincial rates while during the period of observation circulatory disease mortality rates in Essex County were 5% higher than the Provincial average. There is no reason to suspect that there was an association between exposure to asbestos at the Bendix factories and increased mortality from circulatory diseases.

Two deaths in the cohort may have been due to malignant mesothelioma of the pleura, although both were coded as cancers of the lung in the official Provincial statistics. It was found that lung cancer rates were increased among both the men and the women. The elevation in rates was not "statistically significant", and so might be readily attributable to chance fluctuations in the mortality pattern, although other factors are certainly not precluded. Among the men, the increase occurred 20 or more years after potential first exposure, a pattern that one would expect to see if there were occupational causation. If one removes the 2 possible mesothelioma cases from the statistics for lung cancer, then there were 3 cases among the men versus 4.9 expected prior to 20 years latency and 11 observed versus 7.9 expected 20 or more years after

potential first exposure to asbestos. No association was found, however, between lung cancer risk and employment in an asbestos-using Department or between lung cancer risk and total length of employment.

There was a cluster of 4 deaths from cancer of the larynx among Bendix workers, a rate about 10 times higher than the Provincial average. This cluster is difficult to interpret. None of these men worked in an asbestos-using Department, although 2 of them may have had direct contact with asbestos dust in their respective capacities as millwright and member of the clean-up crew. In addition, lung cancer rates are usually elevated more in asbestos-exposed workforces than are laryngeal cancer rates, the opposite of the situation that was found here.

The risk of asbestos-associated disease was found to be lower in the Bendix workforce than in other asbestos-exposed factory populations studied by the Ministry of Labour, presumably because the average exposure in the Bendix population was quite low. It is concluded that surviving members of this workforce are at relatively low risk of asbestos-associated disease. It is standard practice for the Health Studies Service to recommend that all asbestos-exposed workers stop smoking because of evidence that cigarette smoking increases the asbestos hazard and other evidence that the risk lessens following smoking cessation. Since cigarette smoking is also associated with an increased risk of circulatory diseases, a cause of death found to be in excess in the Bendix workforce, smoking cessation would be a valuable health protection measure for Bendix workers.

INTRODUCTION

The Ontario Ministry of Labour has carried out an analysis of the mortality pattern of former employees of the Bendix Automotive Corporation in Windsor in response to concerns, expressed in a letter to the Minister of Labour by the United Auto Workers (now CAW), that the health of employees had been damaged by exposure to asbestos at the Argyle Road and Prince Road factories. Only a small part of the workforce had been directly involved in the manufacture of automotive braking systems using asbestos-containing friction materials, but employees believed that the workforce as a whole had been exposed to fibres of chrysotile asbestos because of dissemination of asbestos from the Departments directly involved with the friction materials. International experience has shown that exposure to asbestos may cause an increased risk of death from cancer and from lung disease. The aim of this study was to investigate whether the exposures at the Windsor factories were associated with an increased risk of death from asbestos-related diseases.

This report presents the results of a study of mortality among 1657 workers who were employed at Bendix for one year or more. Deaths occurring between January 1, 1950 and December 31, 1985 were ascertained by a computerized linkage with the Canadian Mortality Data Base at Statistics Canada and form the basis for the analysis.

DESCRIPTION OF THE FACTORIES

Bendix Automotive of Canada Limited (BAC) began manufacturing operations in Windsor in 1929 when it purchased property on Argyle Road(1). Operations at that time included automatic screw machine work and the manufacture of hydrovacs (the forerunner of the modern power brake assembly unit), wheel cylinders, master cylinders, other automotive component parts, and bicycle brakes. This manufacturing operation continued until approximately 1937 when the manufacture of drum brake assemblies was introduced. BAC purchased brake shoes and linings from the American Corporation for assembly at the Argyle Road plant. These components had already been drilled, ground, and riveted prior to arrival at the Windsor plant.

In 1940 BAC purchased drilling, grinding, and riveting equipment to manufacture its own lined brake shoes, and it is from this time that employees may have been exposed to asbestos-containing dusts. The type of asbestos fibre contained in the brake linings was chrysotile asbestos and the asbestos content generally varied from 25% to 50% depending upon the particular use for the product. The balance of the linings consisted of a resin binder and various modifiers and fillers. The brake assemblies were for Canadian automobile manufacturers only and the process represented a small part of the entire manufacturing operation at the Argyle Road plant. The operation was limited to one shift per day and frequently less than 5 shifts per week. Over 90% of manufacturing operations were devoted to automatic screw machine work and to the manufacture of other automotive components for the war effort, and none of these involved the use or handling of asbestos.

The drilling, grinding, riveting, and brake assembly operations comprised less than 10% of the total manufacturing operations at the plant and were performed in a relatively small area of the Argyle Road plant known, at that time, as

Department 20. Activity continued at this level until 1950 when the operation was relocated to a slightly larger area and the process was modernized by the utilization of a carousel for assembly and a conveyor system to transfer the completed brake assemblies to another area of the plant for shipment. The operation remained in this configuration until 1962 when it was expanded slightly.

In July, 1963 the entire brake assembly operation was transferred to a new BAC plant located on Prince Road, and the assembly operations took place in Departments 25 and 27. A single shift was maintained at the Prince Road facility throughout 1963 and 1964 and into the first months of 1965 when production was increased to two shifts per day. The brake assembly department employed approximately 30 workers out of a total workforce of about 450. In 1969 the drilling, grinding, and riveting operations (Department 25) were returned to the Argyle Road plant while drum brake assembly (Department 27) remained at Prince Road.

The drilling, grinding, and riveting procedures were carried on at the Argyle Road plant from 1969 to April 1980. This operation, formerly known as Department 20 at Argyle Road, became designated as Department 25 upon its return from Prince Road. The drum brake assembly operation (Department 27) remained at the Prince Road plant until June 1980 when the plant was closed permanently.

EXPOSURE TO ASBESTOS

There were only 2 small production areas in the 2 plants that contained friction material products. The 2 departments in question were designated with Department numbers 20, 25, and 27. Departments 20 and 25 were the same, but the number and location were redesignated. Employment in the friction materials departments was relatively low. In 1963-65 only 30 of 450 employees were involved with the friction material

product. The plant reached its highest hourly rated employment level of 778 employees in 1976 at which time only 88 employees worked in friction material operations.

Although workers who were employed in operations involving friction material brake linings containing asbestos had the greatest potential for exposure to asbestos, most of the other workers had potential for exposure to fibres which may have been distributed to other areas of the plant. The level of exposure outside the friction material areas was presumably lower than the levels within them.

It is known that workers were potentially exposed to chrysotile asbestos, present as dust arising from the manipulation of the brake shoes, but there is little quantitative information about actual exposures. The first air sampling survey was performed by governmental hygienists in 1975, and from that time until plant closure in 1980, sampling results, both area and personal showed concentrations of asbestos fibres to be less than the then prevailing standard of 2 fibres per ml of air and concentrations were generally less than 1 fibre per ml. Exposures prior to 1975 are unknown, but may have been higher. It is also possible that individual exposures to employees maintaining the dust collection systems may have been substantially higher than those recorded in the surveys of general work areas.

DEFINITION OF THE STUDY POPULATION

The study cohort consists of all workers who were employed on or after January 1, 1950 for a total period of at least 12 months. The one-year qualification period was chosen to exclude transient workers who would have added little to the assessment of health risk associated with employment at BAC but who would have added substantially to the cost of data collection and follow-up. The entry date of January 1, 1950 was selected because tracing of workers was accomplished primarily through the records of Statistics Canada, which exist in machine-readable form only from 1950 onwards. Exclusion of workers who may have left before 1950 is unlikely to bias the assessment of risk since they could have been exposed to asbestos for a period of at most ten years following the introduction of drilling and grinding in 1940.

DATA COLLECTION

Employee identifying information was abstracted from plant personnel records in Windsor. Shortly before, a California-based research company, SRI International, had been hired by the Bendix Corporation to undertake a parallel study, and SRI personnel had independently copied and coded the same records. By agreement among SRI, Bendix, and the Ministry of Labour the computer files were compared and any discrepancies were traced and corrected.

Collection of information about the jobs performed by each worker was complicated by the fact that detailed personnel folders were not available for all workers. For employees terminating prior to 1968, personnel records had been reduced to a card index system containing only such information as name, address, clock number, and employment dates. A detailed chronology of jobs was not recorded. Sixteen hundred and fifty

seven individuals met the criteria for inclusion in the cohort. Three hundred and forty one of these persons had terminated prior to 1968, while 536 who started prior to 1968 were still employed in 1968 or later. In view of the fact that complete personnel files were not available for all workers it was decided to abstract, for each member of the cohort, only the information available on the index cards.

Detailed work histories were sought later, following ascertainment of mortality, as part of a case-control study of those causes of death potentially associated with exposure to asbestos. The records of individuals participating in the case-control study were copied at the London, Ontario facility of Bendix Heavy Vehicle Systems, where the plant records had been moved following closure of the Windsor operations.

STATISTICAL METHODS

A standard person-years analysis[2] was used to compare mortality in the Bendix cohort with mortality rates in the general population of Ontario. One-sided P-values were computed assuming a Poisson distribution of observed causes of death.

Matched case-control analyses with variable numbers of controls were performed using conditional maximum likelihood methods[3].

RESULTS

A) THE STUDY POPULATION

1) Dates of Hire

The study population consists of 1657 individuals; 1314 males and 343 females. The distribution of the dates of hiring of these workers is given in Table 1. Less than half (701 or 42%) of the 1657 were hired before 1966 and could thus have had 20 or more years pass between the date of hire and the end of observation on December 31, 1985.

2) Length of Employment

Many of the workers in the cohort had breaks in employment because of layoffs. Detailed information about these employment gaps was collected only for the workers in the case-control study, for whom complete work profiles were compiled. As a crude measure of length of employment, suitable for the entire cohort, we have calculated the Gross Length of Employment which is defined as the time between hire and termination. For many workers, Gross Length of Employment is an overestimate of the time actively employed because of breaks due to layoffs. The distribution of Gross Length of Employment in the cohort is given in Table 1. Forty-five percent of the cohort were employed less than 5 years, 17 percent were employed between 5 and 10 years, and 38 percent were employed for 10 years or more.

B) MORTALITY IN THE BENDIX POPULATION

Table 2 gives the results of the comparison of mortality rates in the Bendix cohort with mortality rates in the general population of Ontario. In this Table, the columns labelled "EXP" give the numbers of deaths from each cause that one would have expected to find if the mortality rates among Bendix

workers were the same as the Provincial average. The Standardized Mortality Ratio or SMR is the percentage of the Provincial average mortality that actually occurred. An SMR of 100 thus indicates that the Bendix and Provincial rates were identical, while an SMR greater than 100 indicates higher mortality rates among Bendix workers and an SMR less than 100 indicates lower mortality rates among Bendix workers than in the general population.

Provincial, rather than Essex County, rates were used in the calculation because of the greater statistical stability associated with the rates from the much larger Ontario population. During the period of follow-up Essex County rates differed somewhat from Provincial rates for a number of relevant causes of death[4]. All-Cause Mortality rates were 3% higher in Essex County for both sexes, and the Cancer rate was 3% higher for men, but 3% lower for women. Rates for Lung and Laryngeal cancers were both significantly higher than the Provincial average. For men, the lung cancer rate was 10% above the Provincial average and the laryngeal cancer rate was 28% above the Provincial average; for women in Essex County, the lung cancer rate was 16% above the Provincial average.

Because the health effects of asbestos exposure may appear only many years after the start of exposure, the comparisons in Table 2 have been divided among a number of time intervals to facilitate an examination of the time course of mortality trends in the Bendix workforce. The beginning point for the measurement of "latency" in this study is the time when the worker may first have been exposed to asbestos in the Bendix plants, that is, the date of hire or the date of introduction of brake shoe operations in 1940, whichever came later. The last column in Table 2 presents the data for the period 20 or more years after first exposure, the period when one would expect an "asbestos effect" to be most evident.

The Table presents data for those causes of death usually associated with asbestos exposure, namely lung, laryngeal, and

gastrointestinal cancers as well as non-malignant respiratory diseases. Circulatory diseases have also been included in the Table because they are responsible for approximately half of all deaths in Ontario. The mortality calculations were computed without reference tc the Departments of employment or jobs performed, because details of the work history were available for only a part of the cohort. The results thus give an overall view of the mortality risks faced by the average member of the Bendix population and may be useful for comparison with the risks in other asbestos-exposed workforces in Ontario.

Table 3 presents mortality information organized according to whether the Gross Length of Employment was less than or greater than 10 years. Gross Length of Employment is intended to serve as a surrogate for the cumulative amount of asbestos exposure in the plant environment, but it is recognized that workers with the same length of employment could have quite different personal exposures to asbestos. One would expect that those workers with the greater amount of exposure would experience greater health effects from asbestos.

1) Overall Mortality

Overall mortality rates in the Bendix workforce were increased relative to those of the general population for both men and women. During the period 20 or more years after first exposure there were 104 deaths among the men compared to 95.5 expected. The SMR of 109 is not statistically significant but the extra mortality occurring after 30 years from first exposure is of borderline statistical significance. The excess mortality among the men may be accounted for by increases in fatality from cancer and circulatory diseases.

The women had increased mortality rates during all observation intervals, including the period between 1 and 10 years after first exposure. In the period following 20 years

from first exposure there were 20 deaths compared to an expectation of 11.1. This increased mortality is statistically significant, suggesting that the mortality risks among the Bendix women were truly different from the Provincial average, but the excess among the women was not concentrated among any particular group of diseases. Among the 20 deaths were 6 from tumours, 5 from circulatory disorders, 2 each from diabetes and trauma, and 1 each from infection, and diseases of the respiratory system, kidneys, and nervous system. Among the 6 deaths from tumours were 1 each from lung, skin, breast, ovary, lymphoma, and leukemia.

Table 3 indicates that there was little substantial difference in the mortality patterns of those with a Gross Length of Employment less than 10 years and those with a Gross Length of Employment greater than 10 years, with the exception of deaths from laryngeal cancer. All 4 deaths from laryngeal cancer occurred among men with employment longer than 10 years.

2) Cancer

There were 45 cancer deaths among the men compared with the 40.3 that would have been expected among the general population of Ontario. The extra cancer mortality among the men can be accounted for by increased mortality from lung and laryngeal cancers. These will be considered in more detail below and in the case-control analysis.

There were 9 deaths from cancer among the women compared with the 6.7 that would have been expected among the general population of Ontario. There were 2 deaths from lung cancer compared with an expectation of 0.7; no other anatomical site occurred more than once.

3) Lung Cancer

Eighteen individuals had their causes of death coded to Lung Cancer on the death certificates. In all but one case, additional diagnostic information was available. It was

determined that the diagnosis of lung cancer was based upon findings from X-ray examination (1 case), sputum cytology (1 case), biopsy (4 cases), surgery (4 cases), or autopsy (5 cases). For 2 workers the death was attributed to lung cancer in the Provincial statistics, but the cause of death might actually have been malignant mesothelioma (see discussion below).

3A) Smoking

A telephone survey of smoking habits in the Bendix workforce was carried out by the Ministry of Labour in 1981 as part of an inquiry into the health of the workers. Responses were obtained from 426 individuals, some 25% of the cohort. Information on smoking habit was thus available for only a small proportion of the cohort, and those who replied may, or may not, be representative of those for whom information is not available. Of those who replied, 20% of the 360 men and 26% of the 66 women stated that they had never smoked cigarettes.

4) Malignant Mesothelioma

For 2 workers the cause of death was coded to lung cancer in the Provincial statistics but there is some reason to believe that the actual cause might have been pleural mesothelioma. One of these men began work in 1952 as a machine operator in Department 16 (Hydraulic Assembly). He continued to work in this capacity until 1966 when he underwent chest surgery and had a biopsy of a pleural mass. At surgery the pleural space was found to be diffusely involved with tumour and the pathologist in Windsor diagnosed pleural mesothelioma. Regrettably, the slides from this case have been lost, and it is not possible to obtain a confirmatory opinion from a second pathologist. However, the Pathology Consultant to the Workers' Compensation Board stated that, based on the surgical findings and the description of the tissue, mesothelioma was the most probable diagnosis.

In the second case, the man began work in August, 1968 as a machine operator in Department 28 (Mastervac Assembly) at Prince Road. He subsequently developed a chest tumour and a biopsy was obtained. The pathologists who reviewed the material did not feel able to make a firm diagnosis. The Pathology Consultant to the Workers' Compensation Board expressed the opinion that the morphology of the biopsy was fully consistent with mesothelioma, as was the distribution of the tumour at surgery. He could not, however, exclude the possibility of metastatic carcinoma. This worker died in 1981, less than 13 years after starting at Bendix.

5) Cancer of the Larynx

There were 4 deaths from laryngeal cancer. All occurred in men with more than 10 years of employment and in each case there was a firm histological diagnosis of squamous cell carcinoma. For one man who died in 1976, 36 years after potential first exposure to asbestos, the disease had actually been diagnosed 14 years earlier in 1962.

6) Gastrointestinal Cancer

There was no increase in mortality rates from gastro-intestinal cancer among Bendix workers.

7) Non-Malignant Respiratory Disease

There was no increase in mortality rates from respiratory disease in the cohort. No cases of asbestosis were diagnosed. Some evidence about the general level of asbestos exposure in the plant may be deduced from information obtained by pathologists who examined lung tissue at post-mortem. Asbestos exposure commonly leads to changes of the pleural surfaces of the lungs and chest cavity which manifest themselves as thickenings or adhesions at pathological examination. Among the autopsy reports we were able to obtain were 19 which contained descriptions of the lungs and pleura of Bendix

workers. In no instance was there a mention of fibrosis (scarring) of the lungs. Four reports (all from deaths unrelated to asbestos exposure, namely, trauma, perforated ulcer, gall bladder disease, and congestive heart failure) mentioned pleural abnormalities which were: 1) a few old adhesions; 2) a few fibrous adhesions bilaterally; 3) right pleural cavity normal, left obliterated by dense adhesions; and, 4) focal fibrous adhesions right lung. These observations may be compared with similar information pertaining to workers at a Toronto asbestos-cement factory where asbestos exposure had caused considerable disease[5]. Autopsy reports from 47 asbestos-exposed workers mentioned lung scarring in 29 cases and pleural abnormalities in 27 of 35 cases in which the pleura were commented upon. Among workers at that factory who were believed to have had minimal exposures, 2 of 7 reports mentioned lung scarring and 4 of 5 mentioned pleural abnormalities. Although selection factors preclude a precise comparison between these 2 sets of reports, it may be concluded that the Bendix population, as a whole, had substantially lower exposures to asbestos than did the workers at the Toronto plant.

C) THE CASE-CONTROL ANALYSIS

Because work histories were not available for all members of the cohort, a case-control analysis was undertaken to study associations between occupational exposure factors, such as employment in an asbestos-using Department, and the risk of death from asbestos-associated diseases. An attempt was made to match each individual (the CASES) dying from lung cancer, laryngeal cancer, gastrointestinal cancer, or non-malignant respiratory diseases with 3 other individuals (the CONTROLS) who survived to at least the age of the corresponding case subject. Controls were randomly matched to cases, using gender

and year of birth as matching factors. For some of the older case subjects, it was not possible to find 3 eligible individuals to serve as controls, and for these sets fewer controls were used.

Work records of the subjects in the case-control analysis were sought at the London, Ontario plant where the Windsor files had been sent for storage. The quality of the information available was variable. Records could not be located for 5 individuals (control subjects), and for 8 other persons the file contained only pension data, giving date of hire and years of pensionable service, but no chronology of job locations. For the majority of subjects, however, it was possible to track departmental assignments.

The employment record was coded for the computer, noting the dates of assignment to the various departments. When a specific department could not be identified, only the start and finish dates were noted. Potential exposure to asbestos was measured in 2 ways. Departments 20, 25, and 27 were taken to be the ones in which asbestos had been manipulated and in which there was possibility for direct exposure. The number of days of employment in these departments up to 5 years before the death of the case, or up to 3 years before the diagnosis of laryngeal cancer, was computed for the individuals in each matched set. Potential background exposure to asbestos was assessed by computing the total length of employment after 1939 up to 5 years before the death of the case, or up to 3 years before the diagnosis of laryngeal cancer for the individuals in each matched set.

1) Lung Cancer

Table 4 presents the results for lung cancer. Two (11%) of 18 individuals dying from lung cancer had ever worked in an asbestos-using department; in comparison, 23% of control subjects had worked in an asbestos-using Department. When allowance was made for a minimum 20 year period between first

exposure to asbestos and death, 12 deaths from lung cancer remained and the percentage of exposed cases (17%) was similar to the percentage of exposed controls. When total length of employment anywhere in the plants was used as a measure of exposure, there was no difference in the proportions of cases and controls employed less than or more than 15 years.

The correct method of analysis for matched case-control data is to take account of the matching process. When this was done, using conditional maximum likelihood analysis, the results were similar to those found with the unmatched proportions shown in Table 4. There was no association between the risk of lung cancer and employment in an asbestos-using Department or with length of employment in the factories.

2) Laryngeal Cancer

None of the 4 men with laryngeal cancer had a job in an asbestos-using Department, although some of them may have been in these Departments carrying out other assignments. One of the men had 33 years of service and had worked as a maintenance millwright. One of his tasks had involved maintaining the furnace used for brazing metal. Every 3-6 months he had to remove and replace asbestos powder from the furnace, a job lasting from 1 to 4 days.

Another man had been hired in 1940 and worked as a machine operator and inspector in the hydraulic assembly departments before his death in 1977. A third worker was hired in 1963 and worked as an inspector in the brake cylinder Department. He died in 1979. The fourth worker dying from laryngeal cancer was hired in 1926, was diagnosed with laryngeal cancer in 1962 and died from metastatic disease in 1976. He had been head of the clean-up crew for the last 20 years during a time in which there was dry sweeping of dust.

Analysis of the matched laryngeal cancer case-control subjects indicated no significant association between the length of employment at Bendix and the risk of laryngeal

cancer.

3) Non-Malignant Respiratory Disease

There was no difference in the distribution of exposure factors between those individuals dying of non-malignant respiratory diseases and their control subjects.

DISCUSSION

This report presents an analysis of the mortality pattern among 1657 workers who were employed at the Windsor factories of the Bendix Automotive Corporation for at least 1 year. The major focus of interest was mortality from those diseases associated with exposure to asbestos. Mortality ascertainment covered the period January 1, 1950 through December 31, 1985 making use of the computerized Mortality Data Base maintained by Statistics Canada. The matching process is a probabilistic one, and it is possible that some deaths might have been missed by the procedure, resulting in an underestimate of the SMRs. It has been estimated that any underestimate is likely to be less than 10% of the value of the SMR[5].

Asbestos-containing automotive brake materials had been introduced to the Argyle Road plant in 1937, but drilling, grinding, and riveting operations did not begin until 1940 in an area known as Department 20. In 1963, the entire brake assembly operation was transferred to the new Prince Road plant, and the assembly operations took place in Departments 25 and 27. In 1969 the drilling, grinding, and riveting operations (Department 25) were returned to the Argyle Road plant while drum brake assembly (Department 27) remained at Prince Road. In this study, the asbestos-exposed Departments were thus taken to be Departments 20, 25, and 27.

Bendix employees were of the opinion that exposure to asbestos had occurred throughout the plant. Representatives of the United Auto Workers testified at the hearings of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario that it was their belief that all workers at the plant had been exposed to asbestos through spread of dust from the active Departments. The concern that all workers were exposed to asbestos is addressed in this study in both the cohort mortality and case-control analyses. The

cohort mortality analysis used length of employment, irrespective of Department, as a measure of exposure; it thus measured the average risk of employees at the plant. To assess risk associated with working in the asbestos-exposed Departments, the case-control analysis segment of this study examined employment in the specific asbestos-using Departments as well as general plant employment as risk factors for asbestos-associated diseases.

There is generally a substantial time lag between exposure to asbestos dust and the development of asbestos-associated diseases. To take account of this fact, the statistical analysis divided the observation period into 10 year intervals (following the date of potential first exposure to asbestos in the plants) in order to assess the trends of disease with time. The date of hire, or 1940, the year in which the manipulation of asbestos-containing materials was begun at Argyle Road, was taken as the date of first possible exposure to asbestos.

The results of the cohort analysis of mortality were presented in Tables 2 and 3. Among the men, mortality rates were similar to the Ontario average during the first 30 years from potential first exposure to asbestos. Subsequently, mortality rates were 25% higher than the Ontario rates. Excess mortality during this latter period is attributable to a cluster of 3 deaths from laryngeal cancer and a 30% increase in mortality rates from circulatory diseases.

The mortality pattern among the women is unusual, in that there is no occurrence of the so-called "healthy-worker effect", the common observation that employed populations usually have lower mortality rates than the general population in the first years after the start of employment. The women had elevated mortality rates during all the time intervals under consideration. Overall, there was a small increase in cancer rates, 9 deaths occurred versus 6.7 expected. There were 2 deaths from lung cancer compared with an expectation of 0.7; no other anatomical site occurred more than once. There

was a larger elevation in death rates from circulatory diseases, 10 deaths occurred while 5.7 were expected based on Ontario rates.

Mortality rates from circulatory diseases were elevated in both sexes, and there was no trend with time. Overall, there was a 19% increase in circulatory disease mortality in comparison with Provincial rates while during the period of observation circulatory disease mortality rates in Essex County were 5% higher than the Provincial average[4]. There is no reason to suspect that there was an association between exposure to asbestos at the Bendix factories and increased mortality from circulatory diseases.

Two deaths in the cohort may have been due to malignant mesothelioma of the pleura, although both were coded as cancers of the lung in the official Provincial statistics. In one case, the diagnosis was based upon examination, by a pathologist in Windsor, of tissue obtained at surgery. Regrettably, the slides from this case have been lost, so that our usual method of requesting review of this difficult diagnosis by the Canadian Tumour Reference Centre could not be followed. The diagnosis in the second case was a tentative one, based upon examination of biopsy specimens. There are unusual features to these 2 cases. Neither worker was employed in an asbestos-using Department. One man worked as a machine operator in Department 16 (Hydraulic Assembly) and the other worked as a machine operator in Department 28 (Mastervac Assembly) at Prince Road. The first worker died 15 years, and the second worker died 12 years, after beginning employment at Bendix. Although such short intervals between first exposure to asbestos and death have been found in other cohorts, it is uncommon for the time interval between the start of asbestos exposure and death from mesothelioma to be so brief. Additionally, in most occupational cohorts exposed to asbestos the incidence rate of mesothelioma increases rapidly with latency. Table 8 compares mesothelioma rates in 3 Ontario

asbestos-using factories studied by the Ministry of Labour. There were 2 possible deaths from mesothelioma among 1657 workers employed 1 year or more at the Bendix factories, 31 deaths from mesothelioma among 2188 workers employed 1 year or more at the Johns-Manville factory in West Hill, and 2 deaths from mesothelioma among 107 men employed 1 year or more at the Holmes Insulation factory in Sarnia. It can be seen in this Table that the mesothelioma rates, between 10 and 19 years latency, were the same at Bendix and Johns-Manville. However, while there were no further cases after 15 years latency at Bendix, the rates increased rapidly in the Johns-Manville workforce, such that the rate between 20 and 29 years latency was 10 times the earlier rate and the rate after 29 years latency was 25 times the 10-19 year rate. The Holmes population was much smaller, but again the rate was higher after 20 years latency than before.

Turning attention to lung cancer, it was found that lung cancer rates were increased among both the men and the women. The elevation in rates was not "statistically significant", and so might be readily attributable to chance fluctuations in the mortality pattern, although other factors are certainly not precluded. Among the men, the increase occurred 20 or more years after potential first exposure, a pattern that one would expect to see if there were occupational causation. If one removes the 2 possible mesothelioma cases from the statistics for lung cancer, then there were 3 cases among the men versus 4.9 expected prior to 20 years latency and 11 observed versus 7.9 expected 20 or more years after potential first exposure to asbestos. The case-control analysis (Table 4) found, however, no association between lung cancer risk and employment in an asbestos-using Department or between lung cancer risk and total length of employment.

Might factors other than asbestos be involved? It is well recognized that cigarette smoking is a major risk factor for lung cancer, but inadequate information is available to compare

the smoking habits of the Bendix workers with those of the general population. There is a paucity of information in the scientific literature about health risks among automotive parts manufacturers. Studies of machinists exposed to cutting fluids and abrasives have not found an increase in the risk of lung cancer, but have suggested the possibility of increased risk of gastrointestinal cancers[6,7]. There was no increase in gastrointestinal cancer rates in the Bendix workforce.

There was a cluster of 4 deaths from cancer of the larynx among Bendix workers, a rate about 10 times higher than the Provincial average. This cluster is difficult to interpret. None of these men worked in an asbestos-using Department, although 2 of them may have had direct contact with asbestos dust in their respective capacities as millwright and member of the clean-up crew. It is generally accepted that, in cohorts occupationally exposed to asbestos, the risk of death from laryngeal cancer is less than the risk of death from lung cancer[8]. Table 9 compares the risks of laryngeal cancer with those of lung cancer and mesothelioma in 4 asbestos-using factories studied by the Ministry of Labour. The Bendix experience was anomalous because there were 4 cases of laryngeal cancer among the 1657 workers in the Bendix population, but no deaths from laryngeal cancer among the 2551 workers in the other factory populations, while at the same time the lung cancer rate was elevated by 26% in the Bendix population but was more than doubled in the other workforces.

There are few established causes for laryngeal cancer, and these involve mainly personal factors such as tobacco smoking and the consumption of alcoholic beverages. Might there have been other occupational causes for the laryngeal cancer cluster in this workforce? Asbestos exposure is generally accepted as a cause of laryngeal cancer[8], although this relationship has recently been called into question[12]. A study of laryngeal cancer causes in British Columbia[13] was unable to identify any occupational factors. An Ontario

study[14] of laryngeal cancer found a weak association with asbestos exposure, an association with exposure to foundry fumes and metal dusts, but no association with exposure to nickel. Given the diversity of jobs held by the Bendix employees dying from laryngeal cancer and the statistically small number of cases(4), it is not possible to draw any firm conclusions about occupational causation.

CONCLUSIONS

Mortality rates among Bendix workers, during the period of observation for this study, were higher than the Ontario average, particularly among the women. There was a slight increase in mortality from those causes often associated with exposure to asbestos, namely mesothelioma and lung cancer, and there was a cluster of four deaths from cancer of the larynx. It was not possible, however, to demonstrate an association between either employment in an asbestos-using Department and the risk of asbestos-related disease, or between length of employment at Bendix and the risk of asbestos-associated diseases.

The risk of asbestos-associated disease has been lower in the Bendix workforce than in other asbestos-exposed factory populations studied by the Ministry of Labour, presumably because the average exposure in the Bendix population was quite low. It is concluded that surviving members of this workforce are at relatively low risk of asbestos-associated disease. It is standard practice for the Health Studies Service to recommend that all asbestos-exposed workers stop smoking because of evidence that cigarette smoking increases the asbestos hazard and other evidence that the risk lessens following smoking cessation. Since cigarette smoking is also associated with increased risk of circulatory diseases, causes of death found to be in excess in the Bendix workforce, smoking cessation would be a valuable health protection measure for Bendix workers.

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TABLE 1: THE DISTRIBUTIONS OF DATES OF HIRE AND OF LENGTH OF EMPLOYMENT IN THE COHORT

A: DATES OF HIRE

	MEN	WOMEN	ALL
Hired before 1950	237	58	295
Hired 1950-1965	326	80	406
Hired 1966 or later	751	205	956
ALL	1314	343	1657

B: LENGTH OF EMPLOYMENT

	Gross Length of Employment (Years)				
	1-4	5-9	10-14	15-19	20 OR MORE
Hired before 1950	18	37	25	25	190
Hired 1950-1965	174	47	25	94	66
Hired 1966 or later	557	192	207	0	0
ALL	749	276	257	119	256

TABLE 2
MORTALITY RATIOS ACCORDING TO CAUSE OF DEATH(OFFICIAL CODINGS)
AND TIME FROM FIRST EXPOSURE OR EMPLOYMENT

YEARS SINCE FIRST EMPLOYMENT OR EXPOSURE

		1-9 YEARS			10-19 YEARS			20-29 YEARS			>30 YEARS			TOTAL > 20		
DIAGNOSIS	GROUP	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR
ALL CAUSES	MALES	28	30.6	91	54	51.0	106	36	40.6	89	68	54.9	124	104	95.5	109
ALL CAUSES	FEMALES	8	3.6	225	4	3.3	121	9	4.5	200	11	6.6	167	20	11.1	180
ALL CANCER	MALES	3	5.6	54	13	11.3	115	10	9.1	109	19	14.3	133	29	23.4	124
ALL CANCER	FEMALES	2	1.3	149	1	1.3	78	4	1.8	221	2	2.3	86	6	4.1	146
LUNG CANCER	MALES	1	1.5	66	4	3.4	118	5	2.9	173	6	5.0	120	11	7.9	139
LUNG CANCER	FEMALES	0	0.1	0	1	0.1	981	0	0.2	0	1	0.3	300	1	0.5	192
LARYNX CANCER	MALES	0	0.1	0	1	0.2	612	0	0.1	0	3	0.2	1422	3	0.35	857
GI CANCER	MALES	1	1.2	85	4	2.6	154	2	2.2	91	2	3.2	63	4	5.4	74
GI CANCER	FEMALES	0	0.2	0	0	0.2	0	0	0.3	0	0	0.4	0	0	0.8	0
NON-MALIGNANT DISEASES																
RESPIRATORY	MALES	0	1.1	0	2	2.3	87	2	2.2	91	3	3.7	81	5	5.9	85
RESPIRATORY	FEMALES	0	0.1	0	0	0.1	0	0	0.2	0	1	0.3	333	1	0.5	204
CIRCULATORY	MALES	15	10.8	139	28	23.4	120	16	21.0	76	37	28.2	131	53	49.2	108
CIRCULATORY	FEMALES	4	0.7	595	1	0.8	122	3	1.4	209	2	2.8	72	5	4.2	119

LEGEND: OBS=NUMBER OBSERVED. EXP=NUMBER EXPECTED. GI CANCER includes esophagus, stomach, and bowel.
In the TOTAL > 20 YEARS Column, the underlined SMRs are those for which $P < 0.05$.

TABLE 3
MORTALITY RATIOS ACCORDING TO CAUSE OF DEATH (OFFICIAL CODINGS)
BY GROSS LENGTH OF EMPLOYMENT AND TIME FROM FIRST EXPOSURE

		YEARS SINCE FIRST EMPLOYMENT OR EXPOSURE											
		10-19 YEARS			20-29 YEARS			>30 YEARS			TOTAL > 20		
DIAGNOSIS	GROUP	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR
ALL CAUSES	EMPLOYED <10	24	20.6	117	15	10.6	142	7	8.1	86	22	18.7	118
ALL CAUSES	EMPLOYED >10	34	33.8	100	30	34.5	87	72	53.4	135	102	87.9	116
ALL CANCER	EMPLOYED <10	2	5.4	37	7	2.9	241	2	2.5	80	9	5.4	167
ALL CANCER	EMPLOYED >10	12	7.5	160	7	8.0	88	19	14.2	134	26	22.2	117
LUNG CANCER	EMPLOYED <10	1	1.5	67	3	0.8	375	1	0.7	140	4	1.5	267
LUNG CANCER	EMPLOYED >10	4	2.2	182	2	2.3	87	6	4.6	130	8	6.9	116
LARYNX CANCER	EMPLOYED <10	0	0.06	0	0	0.03	0	0	0.03	0	0	0.06	0
LARYNX CANCER	EMPLOYED >10	1	0.10	1000	0	0.11	0	3	0.19	1600	3	0.30	1000
GI CANCER	EMPLOYED <10	1	1.0	100	1	0.6	167	0	0.5	0	1	1.1	91
GI CANCER	EMPLOYED >10	3	1.8	167	1	1.9	53	2	3.1	65	3	5.0	60
		NON-MALIGNANT DISEASES											
RESPIRATORY	EMPLOYED <10	1	0.9	111	1	0.5	200	1	0.4	250	2	0.9	222
RESPIRATORY	EMPLOYED >10	1	1.6	63	1	1.9	53	3	3.5	86	4	5.4	74
CIRCULATORY	EMPLOYED <10	13	8.3	157	4	4.6	87	3	3.8	79	7	8.4	83
CIRCULATORY	EMPLOYED >10	16	15.9	100	15	17.8	84	36	27.2	132	51	45.0	113

LEGEND: OBS=NUMBER OBSERVED. EXP=NUMBER EXPECTED. GI CANCER includes esophagus, stomach, and bowel.
In the TOTAL > 20 YEARS Column, the underlined SMRs are those for which $P < 0.05$.

TABLE 4: CASE-CONTROL ANALYSIS FOR LUNG CANCER

1) <u>All Latency Periods:</u>

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Lung Cancer	2(11%)	16(89%)
Control Subjects	12(23%)	41(77%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Lung Cancer	10(57%)	8(43%)
Control Subjects	27(51%)	26(49%)

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2) <u>Latency 20 Years or More:</u>

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Lung Cancer	2(17%)	10(83%)
Control Subjects	8(23%)	27(77%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Lung Cancer	4(33%)	8(67%)
Control Subjects	16(46%)	19(54%)

TABLE 5: CASE-CONTROL ANALYSIS FOR LARYNGEAL CANCER

1) <u>All Latency Periods:</u>

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Larynx Cancer	0	4(100%)
Control Subjects	2(16%)	10(84%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Larynx Cancer	1(25%)	3(75%)
Control Subjects	6(50%)	6(50%)

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2) <u>Latency 20 Years or More:</u>

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Larynx Cancer	0	4(100%)
Control Subjects	2(16%)	10(84%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Larynx Cancer	1(25%)	3(75%)
Control Subjects	6(50%)	6(50%)

TABLE 6: CASE-CONTROL ANALYSIS FOR GI CANCER

1) <u>All Latency Periods:</u>

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With GI Cancer	0	10(100%)
Control Subjects	6(22%)	21 (78%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With GI Cancer	8(80%)	2(20%)
Control Subjects	19(70%)	8(30%)

TABLE 7: CASE-CONTROL ANALYSIS FOR RESPIRATORY DISEASE

1) All Latency Periods:

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Respiratory Disease	2(25%)	6(75%)
Control Subjects	3(19%)	16(82%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Respiratory Disease	4(50%)	4(50%)
Control Subjects	10(53%)	9(47%)

2) Latency 20 Years or More:

	EVER WORKED IN EXPOSED DEPARTMENT	NEVER WORKED IN EXPOSED DEPARTMENT
Individuals With Respiratory Disease	1(17%)	5(83%)
Control Subjects	3(20%)	12(80%)

	TOTAL EMPLOYMENT LESS THAN 15 YEARS	TOTAL EMPLOYMENT MORE THAN 15 YEARS
Individuals With Respiratory Disease	2(33%)	4(67%)
Control Subjects	9(60%)	6(40%)

TABLE 8: A COMPARISON OF MESOTHELIOMA RATES IN 3 ONTARIO ASBESTOS FACTORIES

Number of Cases / Person-Years of Observation
(Rate per 1000 Person-Years in Parentheses)

FACTORY:	BENDIX	JOHNS-MANVILLE[9]	HOLMES[10]
LATENCY			
10 - 19	2/10921(0.18)	3/17146(0.18)	0/715
20-29	0/4603 (0)	18/8875(2.0)	2/221(9.0)
30+	0/3085 (0)	10/2164(4.6)	0/6

NOTES:

BENDIX: 1657 Workers Employed 1 Year or More
JOHNS-MANVILLE: 2188 Workers Employed 1 Year or More
HOLMES INSULATION: 107 Workers Exposed to Asbestos 1 year or More

TABLE 9: A COMPARISON OF THE RISK OF DEATH FROM LARYNGEAL, LUNG, AND MESOTHELIOMAL TUMOURS AMONG WORKERS FROM 4 ASBESTOS-USING FACTORIES TEN OR MORE YEARS AFTER FIRST EXPOSURE

	LARYNGEAL CANCER			LUNG CANCER			MESOTHELIOMA
FACTORY:	OBS	EXP	SMR	OBS	EXP	SMR	NUMBER
BENDIX	4	0.52	769	15	11.9	126	2
JOHNS-MANVILLE[9]	0	1.06	0	49	23.3	210	31
HOLMES[10]	0	0.03	0	4	0.5	800	2
DOMTAR[11]	0	0.20	0	6	3.7	162	0